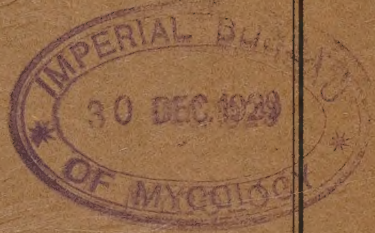
Leaf Scald Disease of Sugar Cane and its Control.

(CONDENSED VERSION)



BY

D. S. NORTH,

Plant Pathologist to
The Colonial Sugar Refining Company Ltd.

Agricultural Report No. 9
(Condensed Version of Agricultural Report No. 8 Technical).

Sydney, March, 1929.

BEGLEY & PIKE LTD., BRISBANE

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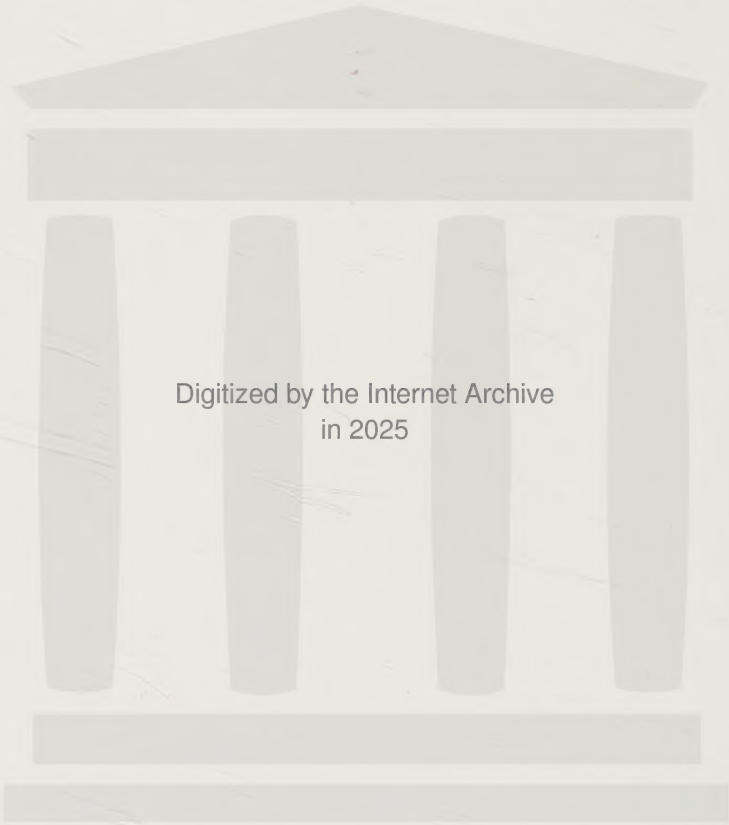
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Leaf Scald Disease of Sugar Cane and its Control.*

(By Mr. D. S. NORTH,
Plant Pathologist to the Colonial Sugar Refining Coy. Ltd.)

Leaf scald is a destructive disease of sugar cane prevalent in Australia and Fiji. What now appears, as the result of Wilbrink's splendid researches (9)†, to be the same disease occurs in Java and was also identified in 1923 in the Philippines by Lee (3, 5). In 1928, Tempany (8) reported its prevalence in Mauritius. In Java it has been known for many years as "gomziekte," being formerly wrongly regarded as identical with Cobb's gumming disease (Bacterium vascularum (Cobb) Greig-Smith). It closely resembles the dreaded "gumming" in many respects, although it is quite distinct, being caused by a different species of bacterium, not yet named.

HISTORY IN AUSTRALIA AND FIJI.

In Australia, its presence has been traced back with certainty to 1911, and it seems likely that it was introduced some time prior to 1900, along with cane plants from either New Guinea or Java. This is strongly suggested by the early history of Mahona (No. 22 of Tryon's 1896 collection of New Guinea canes), which, although a splendid variety in other respects, was said to "mature too early and die off" particularly in tropical districts. It is still liable to "die off" in this manner, but only as the result of leaf scald disease, to which it is one of the most susceptible varieties known.

In these early times, the disease was probably widely distributed through the cane areas of Australia and Fiji, because Mahona apparently failed through "dying off" in practically all districts except the Richmond and Tweed Rivers of New South Wales. In these two districts it yielded highly profitable crops, being widely grown for many years and becoming the main

* A condensed version in more popular language of "Leaf-scald, a bacterial disease of sugar cane," by the same author (7), now out of print. Some minor additions bring it up-to-date.

†Reference is made by number to "Literature cited," p. 48.

Fig. 1.—Typical diseased stalk (chronic form) in one-year-old Mahona. Note the stunted growth, shortened joints, 12 side-shoots and its white chlorotic leaves, curling upwards with withered tips.

standard variety for a time. They apparently escaped infection by some chance. However, leaf scald was introduced from the Clarence to the Richmond in a consignment of 46 tons of N.G.16 cane plants in 1912, and seems also to have been introduced to the Tweed by some unknown means about the same time. In 1915-1917, it became conspicuous in the Mahona crops in both these districts, and during the next few years caused very severe crop losses as will be shown in a later section. On this account the variety finally had to be discarded, or "ran out" as the farmers say, and more resistant kinds grown in its place.

It is likely that leaf scald similarly caused not only Mahona but also other susceptible varieties to "run out" in earlier times in many other districts, but on this point no definite records are available. This is strongly suggested, however, by the fact that the standard varieties now cropped in Australia and Fiji are for the most part highly resistant. Also the planting of Clark's Seedling (H.Q.426), the Gorus (N.G.24, N.G.24A, N.G.24B) and Pompey (7R428) has lately been seriously curtailed in certain districts (e.g. Mossman, Cairns, Johnstone River, Rarawai) owing to its ravages. All of these varieties, too, may be classed as moderately resistant, all being much less susceptible than Mahona.

Hurried surveys made by the writer in 1911 and 1915 have served to establish its prevalence in those times in the Cairns, Johnstone River, Herbert River and Mackay districts. Some suspicious cases of Clark's Seedling suddenly dying off, (not definitely diagnosed as leaf scald, however) were also observed in the Childers district.

PRESENT DISTRIBUTION AND VARIETIES AFFECTED.

A comprehensive view of its present distribution in Queensland has been provided by field surveys commenced in 1924 by the pathological staff of the Queensland Government Bureau of Sugar Experiment Stations (1). These followed the pointing out by McBryde (4) of the serious extent to which the Mulgrave Mill crops, especially those of Clark's Seedling, were suffering from leaf scald. More exhaustive surveys are also made annually of the areas supplying the C.S.R. Co's mills in Queensland, N.S.W. and Fiji, at which technical field officers are stationed for cane

Fig. 2.—Two stools of one-year-old Mahona, in which three stalks (in front) show chronic form of disease (like Fig 1), two small stalks show the acute form, having dead tops, and four tall stalks (behind) are of normal healthy appearance. There are also numerous small diseased suckers springing from the ground.

A B C

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disease control purposes (2). The distribution thus disclosed may be summarised as follows:—

The disease is widespread and causing serious losses almost throughout the Mossman, Cairns and Johnstone River districts (the most northerly) in Queensland. It is now of rare occurrence in the Herbert River district, where it was formerly widespread. It has not been recorded recently in the Burdekin River, Giru or Mackay districts, but occurs to a slight extent at Proserpine which lies between them. Its former occurrence at Mackay has already been mentioned. Only an isolated case has been recorded at Moreton (by Cottrell-Dormer) and again at Bundaberg (by McBryde) both in 1924; and there is no record of it in the Isis, Maryborough, Bauple or Beenleigh districts, although it occurs to a slight extent at Nambour.

In New South Wales, it is still widespread almost throughout the Tweed River district; it is now restricted to a small outlying area on the Richmond. and has become almost extinct on the Clarence, where, although annual surveys have been made, only one isolated case has been detected since 1926.

In Fiji, it was prevalent at Rarawai, where crops of Pompey were seriously damaged and new seedling and other varieties were frequently attacked in the experimental plots, but it is now coming more under control. A slight outbreak has also occurred at Nausori, but there is no record of it from either Lautoka or Labasa, where, however, exhaustive disease surveys have not been recently carried out.

Its greater prevalence in the wetter, as compared to the drier, climates is a striking feature of this distribution, suggesting that wet weather conditions perhaps aid in its dissemination.

However, a more potent factor in this distribution is the fact that Badila acts as a tolerant carrier. Almost every field of Badila has been found to be infected throughout the Mossman, Cairns, Johnstone River and Tweed River districts, in which it is the chief standard variety cropped. Yet appreciable crop damage in this variety is a rare occurrence. Slight, although unmistakable, symptoms are regularly observed in an odd stalk here and there, chiefly streaks in the leaves, and after a time these symptoms disappear again, leaving the stalks apparently healthy, or sometimes slightly stunted in growth. More

Fig. 3.—Another stool of one-year-old Mahona, more advanced stage of disease than Fig. 2. Only one stalk of normal healthy appearance remains.

severe symptoms are observed in rare cases, sometimes odd stalks or even whole stools being killed outright, mostly in crops which suffer a severe check from drought, flood or other cause.

In these districts no economical means of control can be suggested, short of finding another variety equal or superior to Badila and more resistant to the

Fig. 4.—Base of the stool shown in Fig. 3. The small suckers, like the side shoots, exhibit withered leaves, leaf streaks and other symptoms of the disease.

disease. For with such fugitive symptoms, seed selection is impracticable. Small seed beds planted with disease-free Badila introduced from an uninfected locality, rapidly become infected, as was found on the Tweed. And any such measures as introducing disease-free seed cane for whole plantings, or replacing Badila with any other known variety, would, in most areas,

prove more costly than allowing the disease to remain. It therefore seems inevitable that leaf scald will continue widespread in these districts for many years, until the plant breeders find new resistant varieties suitable for replacing Badila. In the meantime, the cropping of the lands unsuited to Badila in these districts with less tolerant varieties such as Clark's Seedling, Pompey and the Gorus, will continue to be fraught with grave risk of infection and crop damage. in spite of every care in seed selection and other measures. On the Tweed it is significant that infection is still widespread in the Badila crops, although more susceptible varieties have not been grown there for several years past, nor have any appreciable crop losses been caused.

However, it is extraordinary that Badila does not apparently behave as a carrier in the Herbert River district, where, although it is the leading variety, and the climate is similar to that of the Cairns district, leaf scald has proved readily controllable by seed selection, combined with some restriction in the planting of the less resistant varieties (Clark's Seedling, the Gorus, Nanemo, Oramboo, Korpi). The same remarks also apply to the upper river areas of the Clarence, and some other localities where Badila is widely grown.

Besides Clark's Seedling, Pompey, the Gorus and Badila in which most of the losses in North Queensland occur, the disease has also been recorded recently in M.Q.1, H.Q.114, Nanemo, Oramboo, Korpi, D.1135, Mahona, N.G.16, Malabar, and a large number of new varieties on trial at Rarawai.

It is also noteworthy that N.G.16, which was at one period fairly generally infected on the Richmond, and on the Clarence to a less extent, and which is a variety considerably less tolerant than Badila, has not served as a carrier in these districts as was at one time feared; it is still widely grown but apparently healthy, having been readily cleaned up since Mahona was eliminated from the surrounding crops.

RESISTANCE OF VARIETIES.

In Table 1 an attempt has been made to place the varieties in order of their relative susceptibility to leaf scald. They are also divided into three groups, viz:—susceptible, moderately resistant, and highly resistant. However, this list must be regarded as a

Fig. 5.—Upper portions of a diseased and an apparently healthy stalk of one-year-old Mahona from adjoining stools, taken in December.

provisional one only and subject to revision. Although based upon all available data, too few observations have yet been made regarding several of the varieties named (as indicated in the table). Also a given variety sometimes seems less resistant in one district than in another, or even on different classes of land in the same district. Thus Clark's Seedling seems highly susceptible in the Mossman, Cairns and Johnstone River districts but moderately resistant on the Herbert River and the Northern Rivers of New South Wales. For this reason, relative resistance needs to be determined to some extent in each individual district.

No immune variety is known, occasional diseased stools having been encountered in every variety which has been observed constantly exposed to infection. Varieties which have not been thus observed as far as the writer is aware (Rose Bamboo, B.208, etc.) have been omitted from the table.

Diseased stools have also been recorded in a large number of little known varieties undergoing trial at Rarawai, Broadwater and other centres. These also have not been listed. Although, as already stated, the present standard varieties are, for the most part, highly resistant, a much larger proportion of susceptible varieties are regularly found amongst newly imported canes and newly raised seedlings.

TABLE 1.—RELATIVE SUSCEPTIBILITY OF CANE VARIETIES TO LEAF SCALD.

(What are considered the more susceptible varieties are named first in order throughout).

Susceptibility.	Variety.	Extent to which observed.
Susceptible	Mahona	Extensively in many districts.
	H.Q.243	Tweed River (N.S.W.) only.
	7.R.96	Few experimental plots, Richmond and Tweed Rivers (N.S.W.).
	8.R.31	Experimental plots, Richmond River (N.S.W.) and Rarawai (Fiji).
	H.Q.114	Goondi Mill (Qld.) only.
Moderately resistant	Nanemo	Many fields, Herbert River (Qld.) only.
	Oramboo	Many fields, Herbert River (Qld.) only.
	Korpi	Many fields, Herbert River (Qld.) only.
	Clark's Seedling (H.Q.426)	Extensively in North Queensland, and few fields, Richmond R.
	Pompey (7.R.428)	Extensively Rarawai, North Queensland and Richmond River.
	Goru and Sports (N.G.24, 24A, 24B)	Extensively in North Queensland.
	N.G.16	Extensively on Northern Rivers of New South Wales.
	M.1900 Seedling	Several fields, Richmond River.
Susceptible but tolerant	Badila	Extensively in many districts.
Highly resistant	Malabar	Extensively on Richmond River.
	N.G.14	Several fields, Richmond River.
	Innes 131	Extensively on Richmond River.
	D.1135	Extensively on Richmond River.
	H.109	Extensively at Rarawai.
	H.Q.5	Many fields on Richmond River.
	H.Q.409	Extensively, Herbert River and Rarawai.
	Q.813	Extensively, Herbert River and Tweed River.

LOSSES.

Stools of cane are frequently killed outright by leaf scald. In the most severe cases, it thus renders the crops of whole fields worthless. But more usually crops are only partially destroyed and many slightly affected crops suffer no appreciable loss.

Detailed estimates of the crop losses due to leaf scald were made in the Mahona crops on the Richmond River harvested in 1920 to 1923. A total of about 750 acres of Mahona were harvested in each of these years. The area affected and tons cane lost are summarised in Table 2.

TABLE 2.—ESTIMATED LOSSES (TONS CANE) FROM LEAF SCALD FOR FOUR SEASONS ACCORDING TO AREA (ACRES) OF DISEASED MAHONA HARVESTED.

	Diseased Areas Harvested (acres)			
Season	Severely Affected	Slightly Affected	Total	Total Losses Tons Cane.
1920	65	360	425	940
1921	132	228	360	2,017
1922	235	223	458	2,179
1923	320	131	451	2,570
Total, 4 seasons	752	942	1,694	7,706

Nearly all these losses occurred in the 752 acres classed as " severely affected." Of these, 31 acres were a total loss, not being worth harvesting, and accounted for 535 tons cane estimated loss. Losses ranged from 25 per cent. of the crop upwards on a further 378 acres and from about 10 per cent. to 25 per cent. on the remaining 343 acres.

In addition, the disease caused extensive areas to be ploughed out after harvesting, instead of being ratooned, considerable further losses thus being suffered. Still further losses are attributable to a serious reduction in quality in the diseased canes sent in to the mill, but detailed estimates of the extent of these losses were not made.

But the loss to the district of such a valuable variety as Mahona has proved to be, probably constitutes the most serious loss of all.

Less extensive losses have also been sustained in N.G.16 variety which is considerably more resistant than Mahona. These occurred chiefly prior to 1920 when detailed estimates of losses were not made. In 1920, 11 acres showed a loss of between 5 per cent.

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and 10 per cent. of the crop, but no large area of this variety has suffered severely since then, although it is still extensively grown.

The losses quoted above will serve as an example of the losses liable to be suffered in any single mill district where a susceptible variety is extensively cropped.

DESCRIPTION OF THE DISEASE.

The disease occurs in two distinct phases, a chronic form and an acute form. The two phases are so different in appearance that they were regarded as two separate diseases until late in 1919, when close investigation showed them to be the same. Either phase is frequently seen without the other, but more often the one grades into the other.

Chronic Form.—In affected crops an odd stalk here and there is stunted in growth, has shortened, stiff, upward curling leaves, with withered leaf tips, some leaves being white (chlorotic), and side-shoots sprouting from every eye of the stem. Straight narrow whitish leaf-streaks are to be found, many, but not all, of which run the full length of the leaf blade and leaf sheath. These features are illustrated in Figs. 1-13 and in Plates 1 and 2. Frequently only one, two or three stalks of a stool are thus affected, the remainder being of normal healthy appearance.

The leaf streaks provide the best means for recognition of the disease and should always be looked for when definite determination is required. In newly attacked canes, a single white leaf streak on one leaf is frequently the only evidence of the disease (Fig. 10, A). However, the first sign of disease in older canes (e.g., one-year-old) is sometimes the sprouting of a single side-shoot, followed by two or three more from the eyes next above and below on the same side of the stalk, the intervening eyes on the opposite side of the stalk remaining dormant until later. But this development is soon followed by leaf streaks, which more often precede the side-shoots.

In young cane the leaf streaks are usually clear and well defined, other symptoms being slow to develop, except that severely diseased shoots without discernable leaf streaks, like those illustrated in Fig. 10, B and Fig. 12, frequently arise from diseased cuttings or from stools diseased before harvesting and ratooning. But in older cane, the leaf streaks often become obscured by

Fig. 6.—Upper portion of a diseased stalk of Mahona taken end of August. Two or three months later it would have been like that shown in Fig 5.

partial withering of the leaves of the main stem, when streaks should be searched for on the leaves of the side-shoots or small suckers. Prior to withering the streaks become broader and more diffused, losing their sharply defined even margins. They then gradually develop into broad withered streaks, usually commencing near the leaf-tip and spreading downwards (Plate 1—see coloured inset—and Fig. 9).

The leaf streaks become plainly visible as soon as a young leaf unfolds and turns green. Some may even be detected in the white immature leaves when unfolded by hand. Their remarkable straightness and evenness is due to their following strictly in the course of the leaf veins.

Acute Form.—The disease may also take the form of an acute wilt, particularly in dry weather in crops approaching maturity. Many whole stools and parts of other stools, sometimes even whole fields, may suddenly wither and die in a most alarming fashion. This wilting takes place just as if the canes attacked were suddenly deprived of their roots, neighbouring unaffected canes meanwhile remaining green and of vigorous healthy appearance. The effect on the crops resembles that caused by the Queensland cane grub, so much so that farmers in North Queensland sometimes attribute to grubs damage which is really caused by leaf scald.

As the wilted canes show no distinctive signs of the disease, in order to determine the cause of their death, canes affected by the chronic form must be searched for amongst them. These may be readily found in some cases, or at any rate some small suckers or side-shoots bearing leaf streaks may be discovered near the bases of the stools. But in extreme cases, it may be necessary to wait until fresh young shoots spring from the diseased stools after harvesting and ratooning. For this wholesale wilting sometimes occurs in crops in which previously very few stalks showed signs of the chronic form of the disease; the stools liable to wilt include not only those previously containing a diseased stalk but also many other stools of normal healthy appearance in the vicinity.

This wholesale wilting occurs chiefly in dry spring and early summer seasons in nearly mature crops on the Northern Rivers of N.S.W., but may scarcely be seen at all in seasons of well distributed rainfall. (When it is most liable to occur in North Queens-

land has not been closely observed). On this account, the disease was at first thought to be of intermittent seasonal occurrence; whereas it occurs at all times in the chronic form, and serious crop losses may even be caused without the acute wilting phase appearing.

Fig. 7.—Upper portion of a stalk of Mahona, in a less advanced stage of disease than Figs. 5 and 6, taken in December.

Internal Symptoms.—On cutting open stalks affected by the chronic form, considerable reddening is found throughout their entire length, in the form of fine bright red streaks, which close examination shows to be reddened fibres (vascular bundles). Many of the red streaks are very minute being much finer

than the fibres containing them. Most are seen in the nodes where the fibres branch and knot together. But some also extend into the internodes. They can be seen most plainly in the mature lower portions of the stalk, but they are also visible in the young

Fig. 8.—A large side-shoot of Mahona. The youngest leaves are white and chlorotic, the other leaves being largely withered. Leaf streaks show on the unwithered parts of leaf blades and sheaths.

developing tissues of the cane top and of the side-shoots, even those just commencing to sprout (Plate 2, A).

Similar reddened fibres also occur in the leaf sheaths (Plate 2, A) and to a less extent in the leaf blades (Plate 1).

Stalks killed by the acute wilt, however, have no reddened fibres nor other distinctive internal signs. Their tissues appear perfectly normal in every respect. Even when examined long after wilting, their tissues are merely more or less dry and shrivelled like those of stale healthy cut stalks. The only exception is the case of wilted stalks which have also produced side-shoots or have other symptoms of the chronic form, but these must be regarded as being attacked by the chronic form as well. Wilted stalks frequently have one or more side-shoots sprouting at their butts, with reddened fibres in the nodes from which the side-shoots have sprung.

CAUSE.

Extremely minute bacteria may be seen by microscopic examination of the leaf streaks or of the reddened fibres in the stem. They occur in countless numbers in the sap vessels of the fibres (Fig. 18).

As a rule the bacteria are strictly confined to the sap vessels, as seen in Fig. 18. But their attack is more severe at certain spots, where they break down the vessels, and invade and destroy the surrounding cells, producing smaller or larger cavities. The beginnings of two such cavities in the stem are shown in Plate 2, A, where they appear as red patches. And the microscopic appearance of a cavity in a leaf sheath is shown in Figs. 15, 16 and 17. This cavity, which was nearly a foot long, formed inside a large pronounced leaf streak and caused it to split open for several inches on the inner surface of the leaf sheath, thus allowing the bacteria to escape between the leaf sheaths closely wrapped round the shoot. It was an immature soft white leaf sheath from a small shoot of Mahona, two-month-old ratoons. The shoot was of normal healthy appearance, except for this one leaf streak extending throughout the blade and sheath of one of the leaves, being affected like shoot 3 of Fig. 10, A.

The appearance of the bacteria in the cane tissues when magnified 1000 diameters is shown in Figs. 14 and 20. The relative sizes of the bacteria of gumming and of leaf scald when stained in the same manner are compared in Fig. 14. That their apparent size varies with the method of staining may be seen by comparing B, C and D in Fig. 20.

The red colouring of the fibres is usually accompanied by " wound-gum," a resin-like substance with

Fig. 9.—Lower half of a 6th leaf, showing, on the midrib, a large white streak; just to right of midrib, a white streak withered towards the top; to left of midrib, a fine white streak; and near the left hand edge, a broad withered streak.

which the affected sap vessels of the fibres are blocked in parts. Some parts of these sap vessels contain only bacteria, other parts only wound-gum, and still other parts wound-gum with bacteria embedded in it. Wound-gum, more or less red-stained, is found in connection with many other diseases and injuries from various causes (e.g., borer tunnellings); it is apparently produced merely as the result of the dying of the tissues concerned.

It has been definitely proved that these bacteria are the direct cause of leaf scald, by isolating them from diseased cane tissues, growing them in pure cultures in the laboratory, and then using these cultures to inoculate healthy canes. These canes developed all the usual symptoms of the disease, purely as the result of their inoculation. Examples of these are illustrated in Figs. 10, 11 and 12. The bacteria were again found in their tissues, at points far removed from the site of inoculation, re-isolated in pure cultures and successfully used for the inoculation of further canes.

Artificial Cultures.—The bacteria proved unusually difficult to cultivate at first, owing to the fact that they cannot be successfully cultivated in the media ordinarily employed for this purpose. Ordinary beef broth, beef extract agar, cane juice and various other decoctions prepared from cane, potato slices and potato agar all proved unsuited, as well as a great variety of other media tried. Culture media specially adapted to their peculiar requirements therefore had to be devised. Good results were finally obtained by the addition of uncooked blood-serum to certain agar media, the best used being blood-serum sucrose potato agar and blood-serum 25%-cane-juice peptone agar. Sucrose potato juice and 25%-cane-juice peptone water also proved useful as fluid media.

But the saccharose peptone agar medium used by Wilbrink (9) (sucrose 2%, peptone 0.5%, di-potassium phosphate 0.05%, magnesium sulphate 0.025%, agar 1.5%, pH 7.0) has since proved far superior to any of the above, and can now be recommended as the best for ordinary purposes.

By its use, the bacteria can be readily isolated in pure culture by the usual methods, from either the leaf streaks or the red fibres in the stem. The organism is a slow grower however; single cell colonies usually take 5-7 days to develop to visible size in

Wilbrink's agar, and 7 days or longer, up to 20 days, in other media, at 27°C, which is about the optimum temperature. The bacteria grown in artificial media differ somewhat in appearance from the normal forms found in the cane tissues, the difference in Wilbrink's agar being hardly appreciable, but it becomes very pronounced in some of the less favourable media, in which much enlarged and curiously curved forms are

Fig. 10.—Diseased Mahona plants three months old, resulting from the inoculation of healthy cuttings with a pure culture of the organism.

The main shoot 3 of plant A is just showing the first symptoms of disease, viz.: a fine white leaf streak on the 4th leaf (left side, upper leaf). On the other hand, the two shoots of plant B appeared above ground with white chlorotic leaves; their growth stagnated and they died out when 4 months old. Shoot B1 appeared a week before shoot A3, and shoot B2 a little later.

produced (Figs. 21 and 22). The other details of cultural characters and morphology described by Wilbrink (9) and North (7) are not repeated here.

INOCULATION EXPERIMENTS.

1. Inoculation of Cuttings.—Healthy cuttings, Mahona variety, were inoculated by dropping some of the bacteria from pure cultures on to the cut ends

just before planting. In each test, the alternate cuttings from each stalk were inoculated, the rest being planted as controls.

In the successful tests, the young shoots from about 25% of the inoculated cuttings showed symptoms of the disease when they appeared above ground, as in Fig. 10, B and in Fig. 12. Most of the rest, although of healthy appearance at first, soon developed symptoms, as in Fig. 10, A, about 80% thus showing the disease within four months from planting, and a further small proportion up to 12 months old. Yet the control plants grew in a normal healthy manner throughout this period.

In one test, in which the bacteria used for inoculation had been maintained 10-11 months in artificial culture, no definite leaf scald symptoms developed in any of the inoculated cuttings. Evidently these bacteria had lost their virulence.

In another test in which planting was followed by a period of drought which caused about one-half of the cuttings to fail to germinate, only about 12% of the inoculated cuttings which grew developed symptoms of the disease. The dry conditions evidently also interfered with the development of the bacteria in the cuttings.

Inoculation by dropping the bacteria on to the buds and leaf scars at the nodes of cuttings also proved successful. But only two cuttings were tested by this method.

2. Inoculation of Growing Plants.—Young (2-5 months old) growing shoots of Mahona were inoculated by injecting small quantities of fluid cultures of the bacteria with a hypodermic syringe, into the cane top near to the growing point. Either the very young leaves just above it or the developing stem tissues just below it were thus inoculated.

In both cases leaf streaks were found 11-28 days later in the young green leaves just unfolding. In the case of leaf inoculations, the leaf streaks were found to have developed upwards and downwards directly from the needle holes made by the hypodermic syringe. Subsequently many more leaves bearing streaks appeared, the plants then gradually developing all the usual symptoms of the disease in severe form. In some cases other stalks of the stools besides the inoculated ones became diseased, as early as 10 weeks after the inoculation in one instance.

Stalks removed and dissected five weeks after inoculation showed the usual internal symptoms of the disease and side shoots were sprouting, the disease having spread throughout the stalks right to the bottom in this brief space of time.

As controls, similar shoots were inoculated in the same manner, but with sterile water or sterile culture fluid. These suffered no considerable ill-effects, but continued to grow in a normal healthy manner throughout.

A B

Fig. 11.—Plant A is another three-months-old diseased Mahona plant, while plant B is a healthy control plant from the inoculation test also illustrated in Fig. 10. The appearance of the disease in young cane in the field is also well illustrated by these two figures, all three types of diseased plants being frequently met with.

3. Unsuccessful Methods of Inoculation.—Repeated attempts have been made to inoculate green leaf blades, leaf sheaths, mature stem tissues of growing canes and roots. Also the bacteria in fluid cultures were poured into cane tops around the spindle of young leaves. But in no instance did the plants thus inoculated develop the disease within a few months.

DISSEMINATION AND CONTROL.

The methods by which the disease is disseminated are:—

1. The use of infected planting material.
2. Knife infection.
3. Spreading of infection from diseased to healthy plants by some other means not yet fully understood.

The combination of these methods enables the disease to spread with great rapidity in crops of susceptibile varieties under ordinary farming conditions. They appear quite sufficient to account for every instance of spreading which has been observed, and no good reason has been found to suspect infection from the soil or from any other source but living diseased cane plants. On the contrary, the causal bacteria are of such a delicate nature that they must soon perish in the soil or almost anywhere else apart from the living plant.

Evidence of infection from the soil has been looked for by extended observations in fields replanted after diseased crops had been ploughed out, and in others where diseased plants had been replaced by healthy ones. Also several experimental plantings have been carried out in which diseased stools were uprooted and immediately replaced with either healthy cuttings or young growing stools. But in no instance did infection thus result. A small proportion of the plants under observation certainly contracted the disease, after some months. But this could not be attributed to infection from the soil, because exactly similar spreading of the disease takes place where the soil has not previously been subjected to infection. In another test, the soil around a young growing stool was heavily inoculated with the bacteria from cultures mixed in water, and again no disease resulted.

The possibility that some common weed or other plant besides sugar cane may be attacked and thus aid in spreading the disease has also been considered, but no such alternative host has yet been discovered. The existence of any such factor appears improbable, because extinction of the disease is apparently taking place in certain localities, where susceptible cane varieties have been discarded and the disease brought under control, as indicated on p. 9.

As the aim of all practical control measures is to prevent, or at least to minimise, dissemination,

close consideration of each of the three methods indicated is called for.

1. Use of Infected Planting Material.—This has undoubtedly been the chief means of dissemination. There can be little doubt that the disease was first brought to Australia and was then distributed far and wide in infected cuttings. That it was introduced to

Fig. 12.—Closer view of plant A in Fig. 11. In shoot 2 all the leaves are almost white. This shoot died six weeks later. In shoot 1 the older leaves are somewhat white, but the four youngest leaves are green and of healthy appearance. Several more apparently healthy leaves unfolded, until the first leaf streak appeared at 4½ months old, more streaks and other symptoms then rapidly following.

the Richmond from the Clarence in 1912 in cuttings of N.G.16 is known. Growers have also propagated the disease on a large scale by using cuttings from infected fields for planting. Even those who are familiar with the disease are still propagating it in this manner, because it is impossible to select only healthy seed cane from an infected crop, even from a very slightly infected one. For many canes which are infected are of normal healthy appearance and devoid of any symptoms which betray their condition. And cuttings from these produce diseased stools of cane.

In order to overcome this difficulty, for purposes of its control on the Richmond, the fields of Mahona from which it was proposed to take seed cane were classified as: (1) safe, (2) unsafe, or (3) diseased. The discovery of a single diseased stalk condemned the whole field as "diseased." Proximity to (within a quarter of a mile or so of) a diseased field, or being raised from doubtful seed, placed the field in the "unsafe" class. Only fields which appeared above suspicion were regarded as "safe." The results obtained over large areas fully justified this classification. Plants from "safe" fields regularly yielded healthy crops, except when planted nearby diseased crops. Of 28 growers who used "unsafe" seed cane in 1921, 20 grew diseased crops and only 8 obtained healthy ones. All who used plants from "diseased" fields grew diseased crops without exception, in spite of the fact that, in most cases, only the apparently healthy normal canes were used and all diseased stools were carefully discarded. Some even cut their seed cane from a portion of the field far removed from the single point at which infection had been noted, but without better results. See also North (6).

A seed selection trial, which also threw much light on this matter, was carried out as follows:—

An attempt was made to produce a healthy crop of Mahona by careful selection of cuttings from a diseased field, followed by eradication of diseased stools as they appeared in the young crop.

Plot 1.—About 1000 cuttings were selected from a well grown one-year-old plant crop, in which only about one diseased stalk to each 12 stools could be detected. Cuttings were taken from stools of perfectly healthy appearance only, the whole stool being rejected if it contained a diseased or suspicious stalk.

Fig. 13.—Pieces of whitened leaves from three-months-old Mahona. In A and B (lower and upper portions of same leaf), a leaf streak to the left of the midrib is discernible only in B, where it has withered. Similarly in C, the position of two leaf streaks is indicated by the withering, they being masked by the whitening lower down.

As stalks with an odd side-shoot or withered leaf tips were regarded as suspicious, about 25% of the stools were thus rejected.

Two months after planting, 12 diseased plants were found and eradicated and 6 more up to the fourth month. No more could be detected during the next three months and it seemed that the disease had been eliminated. But between 7th and 12th month the disease appeared in 15 more stools.

The plot was then harvested and ratooned. In the ratoons, 70 more stools came up diseased and a further 34 diseased stools were found up to $2\frac{1}{2}$ months old when eradication was discontinued. At 12 months old, about 25% of the stools showed the disease.

Fig. 14.—A comparison of the bacteria of gumming and of leaf scald, similarly treated.

A—Gumming bacteria in gum oozed from a cutting.

B—Leaf scald bacteria in a leaf streak.

In the second ratoon crop, owing to dry weather conditions, many of the diseased stools failed to strike while those of healthy appearance grew. On this account only 49 diseased stools were found up to 2 months old.

Plot 2.—About 1000 cuttings were selected from the plant crop in Plot 1 at 11 months old for planting a second plot. The chances of success in this case seemed better, because only one stalk in the part chosen for plants showed disease symptoms (a few

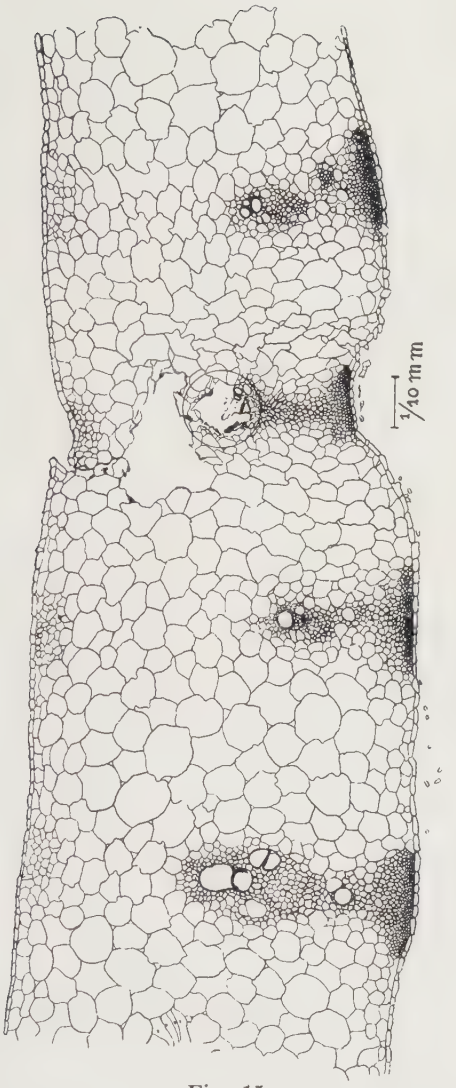

Fig. 15.

Fig. 15.—Transverse section of leaf sheath from a young shoot of Mahona, across a large leaf streak in which a cavity had formed and split open. Here, two cavities have taken the place of a large vascular bundle, which should have been like the one shown on the left, but which has become broken down, much of the debris and bacteria having apparently been discharged through the split, which has closed. The smaller cavity enclosed in a circle is shown more highly magnified in Fig. 16.

leaf streaks) and its stool and that on either side of it were rejected.

However, at 6 weeks old 12 diseased stools and at 3 months old 4 more were found and eradicated. The disease also developed in a few more stools between 7th and 12th months, just as occurred in Plot 1.

In the ratoon crop, 90 diseased stools were again found and eradicated in the first few months, in spite of this crop being affected by the dry weather in the same manner as the 2nd ratoons of Plot 1.

Plots 3 and 4.—Cuttings were selected from the plant crop of Plot 2 at 12 months old, 402 cuttings for Plot 3 and 420 for Plot 4. Plot 3 was planted adjoining Plots 1 and 2, but Plot 4 was located over a quarter mile away and further still from any other cane, with the object of eliminating the possibility of infection from adjoining plots.

Thirty-six diseased stools in Plot 3 and 17 in Plot four were located and destroyed during the first five months; then 78 more in Plot 3 and 21 in Plot 4 were found up to 12 months old.

Drought following planting severely affected both plots, but Plot 4 in light sandy soil, far more than Plot 3 in heavy clay loam. Forty-six cuttings failed in Plot 3 and 61 in Plot 4. The lower proportion of diseased plants in Plot 4, especially in the early stages of growth, was due to the dry conditions rather than to its isolated position, because it was apparent that the disease helped to cause the failure of many cuttings, although exactly how many of the cuttings which failed were diseased could not be determined. Also, growth was slower and the disease slower to develop in Plot 4 than in Plot 3. Therefore there is no reason to doubt that at least all of the plants which developed the disease during the first five months originated from infected cuttings, rather than from infection taking place after planting.

In these two plots the cuttings of each stalk and the stalks of each stool were planted in rotation and the position and origin of each cutting was recorded on plans. By this means it was found that the 53 infected cuttings located during the first five months' growth were not derived from particular stools or stalks, but that 20 of the 26 stools cut each yielded one or more of them. Nine infected out of 49 cuttings was the most from a single stool. Of the 26 stools in

Plot 2, which provided these plants, three developed the disease on being ratooned. These only yielded five, two and one infected cuttings respectively. Individual stalks yielded only one infected out of three to seven cuttings in 26 instances. But pronounced grouping occurred with the other 27 infected cuttings, three consecutive cuttings of the one stalk proving

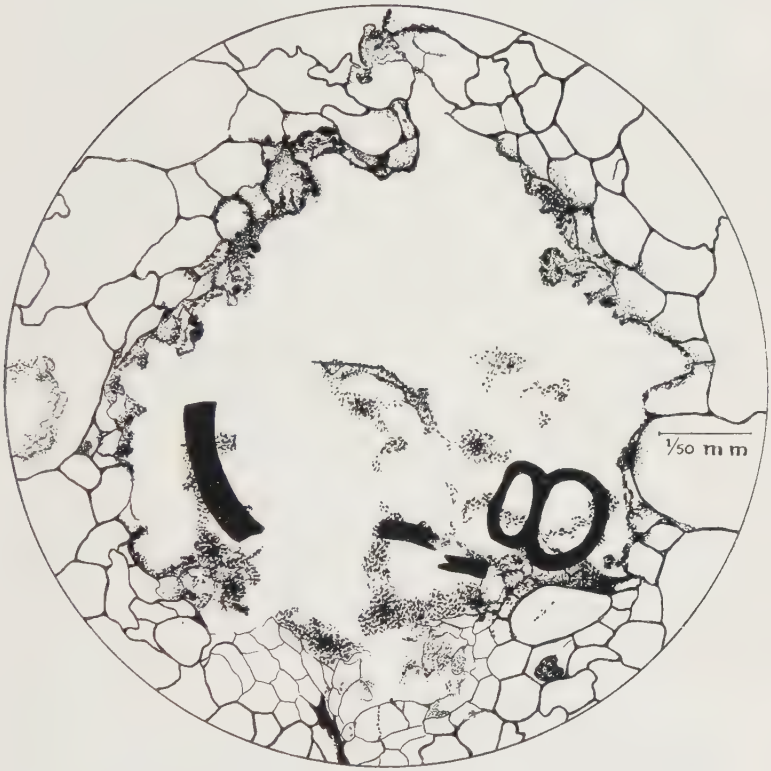

Fig. 16.—The smaller cavity shown in Fig. 15, containing numerous bacteria mixed with the cell debris.

infected in five instances. But in no instance were all the cuttings of one stalk infected. The intermediate and butt cuttings proved more frequently infected than the top cuttings, of which only one was infected.

The distribution of the 99 stools which developed disease between five and 12 months old indicated that many of these also arose from infected cuttings rather than from infection during later development, grouping according to stalks being still more pronounced.

Controls.—420 healthy cuttings from a "safe" source were planted alongside Plot 2 as controls. These all grew without the disease in the plant crop, but 10 diseased stools (2.4%) developed in the young ratoons.

One hundred and fifty cuttings were similarly planted alongside Plot 3 and 180 alongside Plot 4. No leaf scald appeared in these early in the plant crop. but 19 (Plot 3) and four (Plot 4) became diseased when nearly 12 months old, having of course caught the disease by infection spreading from the plots during their growth.

These controls show that the stools in the plots which became diseased before five months old originated from infected cuttings, and that this probably applied also up to 12 months old in Plot 2, which grew in a wet period. But that in Plots 3 and 4, probably half of those which became diseased between five and 12 months old became infected after planting, dry weather having thus caused the disease to show out more quickly.

This trial again shows that it is impossible to select healthy seed cane from an infected crop and that the disease cannot be got rid of by eradicating diseased stools, because many canes carry the infection in a latent, unrecognisable condition.

It also affords a good illustration of the insidious dissemination which takes place, if any infected cuttings be included at planting. The trial also brings out several further important points which will be discussed later.

The result of planting cuttings from stalks of different types from infected crops was also studied by means of planting trials.

It was thus found that **Cuttings from Stalks Affected by the Chronic Phase** often fail to grow. Those which do grow produce weak diseased shoots which soon die, like those shown in Fig. 10, B. Root development is suppressed, the few weak roots which sprout, soon dying off. Even cuttings from stalks showing the earliest recognisable symptom, e.g., a leaf streak on one single leaf, without any buds sprouting, usually fail in this manner. Therefore, the planting of such cuttings of Mahona is of little importance in disseminating the disease, because they mostly eradicate themselves. There is danger with less sensitive varieties, however, as six of 25 similar cuttings

Fig. 17.—Transverse section of the same streak as in Figs. 15 and 16, but 5 inches higher up the leaf sheath and above the split portion. Only the vascular bundle is shown. Here, the cavity is much smaller and confined within the bundle, the remains of the broken down cells being seen lining its walls. There are numerous bacteria amongst the sieve tubes which are being broken down. And the left hand large pitted vessel is blocked with a mass of bacteria embedded in wound gum.

of N.G.16 produced diseased stools which survived till harvested at year old, and three of these again grew as ratoons.

Cuttings from Apparently Healthy Stalks from Diseased Stools usually commence growing in the normal manner. A proportion usually develop disease symptoms, but the majority remain apparently healthy for several months, when symptoms appear in a further proportion, and so on. Similar behaviour was also found for **Cuttings from Apparently Healthy Stools in Infected Blocks,** as shown in the seed selection trial, already described, and in other trials. The only difference was a somewhat higher proportion of diseased cuttings in the former than in the latter. Apparently the proportion infected diminishes more or less as distance from the source of infection increases. All neighbouring blocks of susceptible varieties within range of infection must also be included here, as shown by the results already noted when growers planted " unsafe " seed cane. Also cuttings from an apparently healthy block over 100 yards distant from any diseased cane were planted in one trial and a few proved infected.

Cuttings from Stalks Affected by the Acute Phase.—When " wilted " stalks were carefully selected with buds of normal appearance, many of the cuttings produced apparently healthy stools of cane. This even occurred with stalks of which all the leaves were withered. Only a small proportion developed disease symptoms. They behaved like cuttings from apparently healthy stalks from diseased stools in every respect. This surprising result indicates that the wilting is due to disease of the underground stool without the stalks themselves being directly attacked. When the stalks are also attacked, the fact is indicated by sprouting of the buds. It was found that wilted stalks with sprouting buds fail to grow, or soon die, behaving like stalks affected by the chronic phase alone. Wilted stalks frequently have side-shoots at their base as the disease invades them from underground. For this reason, too, the butt cuttings are the most liable to prove diseased of all the cuttings from wilted stalks with normal buds, and the top cuttings the least liable.

Thus, of the different types of infected planting material discussed, infected cuttings from apparently healthy canes are by far the most dangerous and difficult to deal with from the point of view of dis-

semination and control. That such cuttings are common enough in infected areas to constitute a serious problem in controlling the disease, is evident from the data presented.

In newly infected canes, apparently the parasite is liable to remain for a long period in a latent condition, while the host plant is actively growing. As long as sufficiently uniform thrifty growth is main-

Fig. 18.—Longitudinal section of part of a small-sized vascular bundle in a fine white leaf streak located on the youngest green leaf of a young Mahona shoot. The pitted vessel is blocked up by a mass of bacteria, but none can be seen in the adjoining tissues on either side of it.

tained, development of the parasite may be suppressed. But the latter is ready to commence active development whenever the cane's resistance becomes impaired. This results from any check in growth due to unfavourable conditions, such as dry weather; or by approaching maturity; particularly from preparing and planting cuttings, or ratooning the stools after harvesting, these operations, perhaps, providing the most severe "check" which the cane plant receives in the normal course of its cultivation. It is on this

account that young crops, whether plant or ratoon, are regularly much more diseased than the crops from which they were derived, as was seen in the seed selection trial. Symptoms also regularly show up as the cane approaches maturity in the spring and early summer, especially in dry seasons.

2. Knife Infection.—Experiments were made to determine whether cane knives can act as carriers of infection. Healthy stalks of Mahona from a "safe" source were cut into cuttings with a cane knife with which a diseased stalk had just previously been cut into small pieces. As controls, similar stalks were cut into cuttings with a sterilised cane knife. Several separate tests of this kind were carried out.

The disease developed in about 50% of the stools produced by the knife infected cuttings, while all of the control cuttings produced healthy stools. The proportion of diseased stools and the time of appearance of symptoms was again largely influenced by the conditions of growth, just as already described in the other planting trials.

In view of the wide use made of cane knives in ordinary farm practice, it seemed that knife infection might be one of the chief modes of dissemination. In harvesting crops for the factory, it would serve to transmit the disease from stool to stool in fields to be ratooned. The cane cutters might also carry infection on their knives from one field to another. In harvesting seed cane and preparing cuttings for planting, not only would the ratoon crops be concerned, but the fields newly planted would be still more vitally affected. The seed cane would be liable to knife infection both while being harvested and while being cut up into cuttings.

Knife infection thus appeared likely to offer the chief explanation of the marked increase in the proportion of diseased plants regularly observed in both young plant and young ratoon crops over that of the previous crops from which each was derived. But this was found not to be the case in the planting trials, in which precautions were taken to avoid knife infection but a similar increase in diseased plants still occurred. In the seed selection trial, for example, a freshly sterilised knife was used for cutting each individual stool at harvesting, and for cutting up each stalk in preparing the cuttings for the planting of all

plots except Plot 1, for which the cuttings were prepared without these precautions. But in this trial (as in others), the knife sterilisation apparently had no appreciable effect on the number of diseased stools recorded (pp. 30-34). Evidently some other means of dissemination is mainly responsible for the increases constantly observed. Infection occurs beforehand and remains latent in the canes, until stimulated to activity by the cutting and planting, or ratooning, as indicated above (p. 39).

On the other hand, the use of infected knives for cutting in a disease-free area would be a most serious

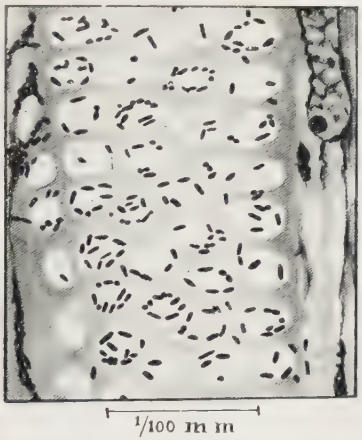

Fig. 19.—Longitudinal section showing portion of the pitted cell wall of a small vessel, with bacteria enmeshed in the pits. This is another part of the vascular bundle shown in Fig. 18

matter, particularly in the case of seed cane. And in all experimental work, constant precautions to avoid knife infection are most necessary.

The sterilisation of knives and other implements may be readily effected by heat or antiseptics in a variety of ways, and is desirable whenever they have been in contact with diseased plants, either known or suspected, before again being used with healthy plants. In the experimental work, usually knives were immersed in boiling water for a few minutes, and chopping blocks were scorched over a naked flame.

3. Spreading by Other Means not yet Fully Understood.—That the disease is rapidly disseminated by some other means besides infected planting material

and knife infection, is abundanty clear from the observations recorded above. Human agencies, such as wounds to roots by implements, may be partly responsible. But that some natural agency, such as a flying insect also plays an important part, is apparent from the frequent spreading of the disease from field to field even though they belong to different farmers and are cultivated by a separate set of implements and stock.

It may be said that fields of Mahona within about a quarter-mile of diseased cane rarely survive a season without an odd stool or two becoming infected. The disease has even appeared in odd cases in fields over half-a-mile distant.

When the disease first appears in a field in this way, it is usual to find either one diseased stool, or two or more widely separated, near the edge or less commonly towards the middle of the field. In the succeeding ratoon crop patches of diseased stools frequently develop around these, "ringworm" patches, as some farmers call them. Odd diseased stools scattered through the fields are usually to be found in addition. In this manner fields of Mahona usually become diseased throughout in the course of two to four years.

Evidence suggesting two chief modes of infection in connection with this spreading has been obtained, viz., "bud infection" and "root infection."

"Bud infection" is suggested by the fact that one or more individual buds on a stalk are frequently infected, while all of its other buds are still healthy. This was well brought out in Plots 3 and 4 of the seed selection trial. A diseased and an apparently healthy shoot springing from different buds of the one cutting has also been noted frequently. Also the sprouting of a single bud as the first symptom of disease in older standing canes, and the dissection of such stalks indicate the bud as the original site of infection.

Bud infection seems to occur in the natural spreading from one field to another over considerable distances and in the infection of scattered stools. The chronic form of the disease primarily develops from it.

"Root infection," on the other hand, is suggested by the rapid wilting of stools in the acute phase, which has every appearance of a root disease, as indicated on pp. 19 to 22 and 38. Even when, owing

to favourable growing weather, this wilting is more or less suppressed, the symptoms of the chronic form first appear near the butts, as the disease travels up the stalks from below. This form of the disease occurs characteristically in the "ringworm" patches, which evidently result from an intense, strictly localised spreading, as if dependent upon the intermingling of the roots of neighbouring stools.

The two methods of dissemination thus suggested appear of prime importance, the most formidable obstacle to successful seed selection being "bud in-

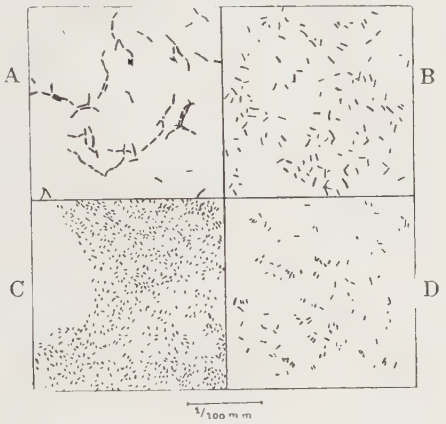

Fig. 20.—Leaf scald bacteria, normal forms:—

A—In a newly isolated culture on blood-serum sucrose potato agar. The bacteria are mostly in chains or pairs.

B—Crushed out from diseased stem tissues and stained on the slide.

C—As seen in a lightly stained section.

D—As seen in a more densely stained section.

fection," while "root infection" apparently originates the acute phase, upon which the disease's destructiveness to crops mainly depends. Further investigation, aiming at the full elucidation of these methods of dissemination, is therefore the most promising line of attack for finding better methods of control.

RESUME OF CONTROL MEASURES.

The control measures already available may now be summed up as follows:—

While no immune varieties have been discovered, many have proved so highly resistant that little further is needed in the way of control measures,

where these are grown and susceptible varieties are eliminated. The comparative resistance of varieties was discussed on pp. 12, 14 and 15.

Where a highly susceptible variety like Mahona is cropped, the most exacting precautions are needed to ensure that no infected cuttings are planted. Owing to the fact that canes of healthy appearance frequently carry the disease in a latent condition, all canes within a quarter-mile or so of diseased plants must be avoided. Classification of fields for this purpose as (1) safe, (2) unsafe, or (3) diseased, on the

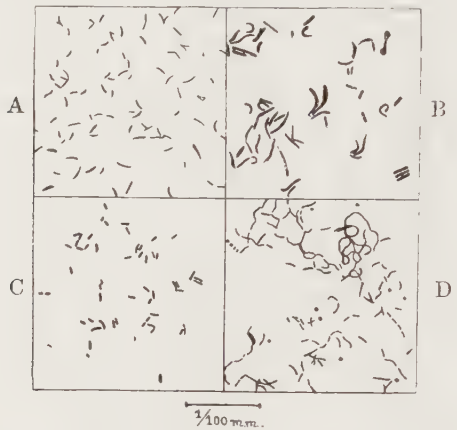

Fig. 21.—Leaf scald bacteria in cultures, involution forms:—

A—Subculture 5 days on sucrose potato agar. Slightly enlarged, thick, curved forms.

B—Subculture 14 days on blood-serum glucose agar. Greatly enlarged, curved and distorted forms.

C—Subculture 12 days on blood-serum sucrose potato agar. Short, thick, slightly curved forms.

D—Subculture 5 days on glucose agar. A variety of shapes, some being much curved.

basis indicated on p. 30 is a good practical system. The sterilisation of infected knives, implements, etc., is also a necessary measure. And, as every diseased cane provides a possible source of infection for all healthy canes within range of infection, all diseased crops should be eradicated as soon as practicable. This usually means the ploughing out of "diseased" crops immediately after harvesting. Eradication of individual diseased stools proves of little value, as indicated in the seed selection trial (pp. 30-36).

Where these precautions are impracticable because the disease is too widespread, the susceptible variety must be replaced.

Where a moderately resistant variety, such as N.G.16, is concerned, control on similar lines becomes

Fig. 22.—Leaf scald bacteria in subculture 5 days on glucose agar. The extreme involution form, consisting of long twisted threads.

a much simpler matter, owing to the slower rate of spreading of the disease in it.

However, with a susceptible but tolerant variety, like Badila, which behaves like a "hidden carrier," control becomes a very difficult problem, as discussed on pp. 12-14.

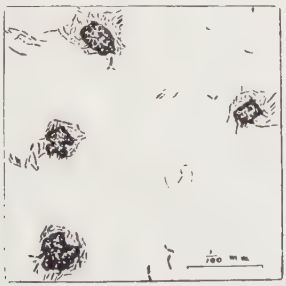

Fig. 23.—Peculiar clumping of bacteria frequently observed in stained films from agar cultures.

SUMMARY.

Leaf scald disease of sugar cane occurs in Australia, Fiji, Java, the Philippines and Mauritius. It seems to have been introduced to Australia prior to the year 1900, and later on to have caused the failure of Mahona in almost every district, except the Richmond and Tweed Rivers of N.S.W., which it did not reach until about 1912. It subsequently caused severe losses in the Mahona crops in these two districts, the losses on the Richmond alone in the four years 1920-23 having been estimated at 7,700 tons cane from an infected area of 1,700 acres. In addition, the crops were reduced in sugar content and many areas could not be ratooned. The variety also had to be discarded.

Its prevalence is now much restricted, owing to the cropping of resistant varieties generally. However, it is still widespread in the Mossman, Cairns and Johnstone River districts of North Queensland and in the Tweed River district of New South Wales, all of which have wet climates and grow mainly Badila, which acts as a tolerant carrier of this disease. In the remaining districts, it has become of rare occurrence, even extinct in a few cases.

Leaf scald is a disease of the vascular system, somewhat resembling gumming, but caused by a different species of bacterium not yet named, exhibiting different symptoms and not producing any gum. The disease occurs in two distinct forms. In one, whole stalks or stools suddenly wilt and die. In the other, white or withered streaks appear in the leaves and numerous side-shoots develop on the stalks, which are stunted in growth and inside which numerous tiny reddened fibres may be seen, particularly in the nodes. The peculiar fine straight white streaks in the leaves provide the best symptom for its recognition. In older cane they may be found more readily in the side-shoots than in the cane top, where they may be obscured by withering. Where the wilting form occurs alone, diagnosis may be difficult or even not immediately possible; small sickly suckers bearing leaf streaks need to be searched for at the base of the stool; these will usually appear, at any rate, after harvesting and ratooning.

The bacteria which cause the disease occur in myriads in the leaf streaks and in the reddened fibres in the stalks, from either of which they may be readily isolated and grown in pure cultures. But special media are needed for this purpose, the best used being Wilbrink's saccharose peptone agar. Proof has been obtained that they are the cause by using pure cultures to inoculate healthy canes, which then developed the disease in its characteristic form. The bacteria were re-isolated from the leaf streaks and stalks of these plants in pure culture and successfully used for the inoculation of further canes. The cut ends of cuttings and the very young leaves and stem tissues close to the growing point in the cane top proved susceptible to inoculation, but the mature leaves, leaf sheaths and stem tissues of growing plants, and roots, did not.

In the field, the disease is disseminated by: (1) The use of infected planting material, (2) Knife infection, and (3) Spreading of infection from diseased to healthy plants by some other means not yet fully understood. Infection from the soil, from other kinds of plants, or from any other source but living diseased cane plants, is believed not to occur. Practical methods of controlling the disease depend upon the prevention of dissemination by these means.

(1) The selection of healthy seed cane is a most difficult matter, because cane of perfectly healthy normal appearance is frequently infected and will give rise to diseased plants if used as seed. On this account, the disease no doubt was first brought to Australia and then distributed far and wide in infected cuttings, and is still being propagated on a large scale by the growers. Extensive field observations and careful tests have proved that, in practice healthy seed cane cannot be selected from even a slightly infected crop. In the case of a susceptible variety, any cane grown within a quarter-mile or so of diseased cane is unsafe.

(2) Healthy canes are liable to become infected by being cut with knives with which diseased canes have previously been cut. This is preventable by knife sterilisation.

(3) It is evident that the disease spreads freely from plant to plant over considerable distances, as if carried by flying insects. There are also some indications that infection takes place in the dormant side-

buds, in which it remains inactive until the buds commence to sprout, as they do when cuttings are prepared and planted, thus arising the difficulty in selecting healthy seed cane.

Another kind of intense local spreading also occurs, as if dependent on the intermingling of the roots of neighbouring stools. It largely accounts for the heavy crop losses frequently experienced.

The disease may be readily controlled by the cropping of resistant varieties. Although no immune varieties are known, several are so highly resistant that no other control measures are needed. And owing to the slower rate of spreading, control may be more readily effected with moderately resistant than with susceptible varieties, with which the most exacting precautions become necessary. Susceptible but tolerant varieties, like Badila, also present a difficult problem.

LITERATURE CITED.

1.—**Cottrell-Dormer, W., N. L. Kelly, E. J. F. Wood:** Monthly Reports to the Director, Bureau of Sugar Experiment Stations; Queensland Agricultural Journal, monthly in Vols. XXIII. to XXVII. (1924-1928) inclusive. Also Annual Reports of the Bureau of Sugar Experiment Stations for 1924-1927 inclusive. Queensland.

2.—**C.S.R. Co.'s Field Chemists:** Departmental Reports to the General Manager, C.S.R. Co. Ltd. (not published).

3.—**Lee, H. A.:** Gum diseases of sugar cane in the Philippines.—Phytopath., XIII, 11, p. 504, 1923.

4.—**McBryde, D. L.:** Circular to Cairns cane growers, May, 1923.

5.—**North, D. S., and H. A. Lee:** Java gum disease of sugar cane identical to leaf scald of Australia.—Phytopath., XIV., 12, p. 587, 1924.

6.—**North, D.S.:** The control of sugar cane diseases in Australia.—C.S.R. Co. Ltd. Agric. Rept. No. 6, Sydney, N.S.W., 1923. (Also in Aust. Sugar Journal, March, April, May, 1923).

7.—**North, D. S.:** Leaf scald; a bacterial disease of sugar cane.—C.S.R. Co. Ltd. Agric. Rept. No. 8, Sydney, N.S.W., 1926.

8.—**Tempany, H.:** Agriculture in Mauritius. Tropical Agriculture V. 9, 1928, p. 227.

9.—**Wilbrink, G.:** De Gomziekte van het Suikerriet, hare Oorzaak en hare Bestrijding.—Archief. Suikerindus, Ned-Indie, 28, pp. 1399-1525, 1920.